June 13, 1958

Dr. Jack L. Strominger
The Edward Mallinckrodt
Department of Pharmacology
Washington University
Euclid Avenue and Kingshighway
Saint Louis, Missouri

Dear Jack:

Forgive me for having delayed so long in answering your letter of March 15 - there are the usual excuses and not much to say. Also, perhaps unfortunately, between the time I last saw you and your letter we got onto some different tasks, but have not altogether abandoned the wall mutant problem.

We have not had much luck still in finding mutants for other metabolites besides DAP. If anything turns up, we'll let you know pdq. We have some new methodological angles to try out.

You have the principal DAP mutants already. W3231 is Davis' 173-25, and essentially equivalent to Work's strain too. W3652 is representative of one of our new mutants; I will dig out some more of the rest. I never have gotten McGilvery's second mutant, have you?

Davis has mentioned that DAP protoplasts became more resistant to lysis with time. But if you prepare them quickly in broth, they are much more fragile. I argue with you of course about Pardee and Trucco.

I hope you will have forgiven my neglect and keep me in touch.

By the way, we have a mutant now (Gal$^{-}_{2}$) which Kalckar says is deficient in the epimerase as well as the first two enzymes. He means to make the obvious inquiry whether galactose is still present in the cell walls. (The mutant does not require galactose for growth after all. Beyond this can you see any application to the wall problem?)

STROMINGER, J.L.

Do you know how glucosamine is metabolized by E. coli? If it goes through UDPGa -- UDPG would it be profitable to look for a glucosamine-negative mutant? Or would we have to feed it UDPGa rather than Ga? Any information on the utilization of UDP conjugates by intact cells?

Yours,

Joshua Lederberg
Professor of Medical Genetics

JL/ew